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The Influence of Exercise on Bone Atrophy

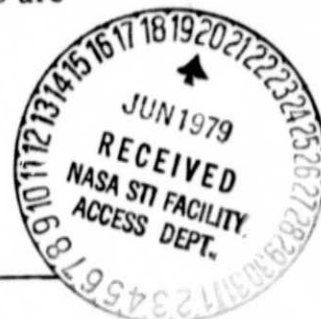
(NASA-CR-160196) THE INFLUENCE OF EXERCISE  
ON BONE ATROPHY (General Electric Co.) 5 p  
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This report describes the relationship between the skeletal system, the muscular system, and exercise in bed rest studies. The regime of exercises performed, the mineral balance data derived, and the bone densitometric data obtained are discussed. A brief review of some of the histological results are also given.



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## THE INFLUENCE OF EXERCISE ON BONE ATROPHY

Bone mineral loss is a phenomenon that has been observed during exposure to weightlessness, immobilization, and extended periods of bed rest. The rate of bone mineral loss in each case differs, but bed rest and immobilization appear to mimic the effects of weightlessness sufficiently well to be used as models for its study. The losses are manifested as increased calcium excretion and decreased bone density roentgenograms when compared to normal, ambulatory values. During these conditions muscle atrophy is known to occur also and is made evident by a loss in muscle mass and muscle strength. Muscle atrophy is known to occur due to lack of use although the actual processes of atrophy are not well understood. Since muscle maintenance is a function of use, of interest is whether the maintenance of bone is dependent upon the same factor. The relationship between the skeletal system and the musculosystem in bed rest is the main focus of this discussion.

Histological evidence of muscle and bone atrophy has mainly focused on the use of rats and monkeys. Hazarian and Von Gierke (1969) presented a histological relationship between muscle and bone atrophy in immobilized *Macacca mulatta* Rhesus monkeys. They found evidence of bone atrophy as a decrease in the size and number of trabeculae and in the thickness of the cortical bone. Furthermore, enhanced bone resorption was found to be occurring at the site of muscle insertion on the bone and periosteal surfaces resulting in a loss of 30% of the force required to pull the tendon from the bone.<sup>2</sup> Monkeys thus far have exhibited similar calcium responses to immobilization as humans so the same type of interaction may be occurring in humans. Muscle and bone are functionally highly interrelated and the compressive and tensile behavior or force of muscle upon bone maintenance has been alluded to also.

Retarding or reversing muscle atrophy is accomplished primarily via exercise. Because of the interaction between skeletal tissue and muscular tissue, exercise is believed to be capable of reversing or retarding bone demineralization as well. Brannan, et al<sup>1</sup> tested separately the effect of isotonic and isometric

exercises upon muscle and bone atrophy for up to 60 minutes/day. Either form of exercise was effective on the arms in terms of muscle strength and muscle mass, but only isotonic exercise with resistance had an influence on thigh muscle atrophy, none on calf muscle atrophy. The study exhibited no loss of bone mineral content during the 60-day recumbency<sup>1</sup> so exercise upon bone loss could not be examined. An exercise regime of both isometric and isotonic exercises upon an individual for 80 minutes a day was evaluated by Hulley, et al<sup>4</sup> with the main emphasis on the isotonic exercises on the legs. The mineral balance data yielded no retardation of calcium, nitrogen, or phosphorus urinary loss during a 24 and 30 week bed rest with exercise study. The mean calcium and phosphorus balances dropped to -267 and -227 mg/day, respectively. A loss of total body calcium occurred at the rate 0.7%/month for the first month and 0.5%/month thereafter. A 10 fold greater rate of density loss was observed in the calcaneus by gamma ray transmission scanning while no losses were detected in the radius or ulna during both the untreated and exercise treated periods of bed rest. These results indicate that the exercise regime had no effect upon bone atrophy during bed rest.

The exercise also had no apparent effect upon the hormones circulating in the blood as plasma calcium regulators. The levels of parathyroid hormone and calcitonin did not appear to change between bed rest untreated and treated subjects, but the sensitivity of the assay procedures may not have been adequate. Vitamin D and its metabolites were not studied.

The design of the exercise program was to attempt to duplicate the compressional and tensile forces applied to the bones by the force of gravity and anti-gravitational muscles. The exercises provided the muscles with a daily regime of movement, but not much force. The pressure forces of the exercise apparatus were only 8 lbs. for less than 80 minutes a day. This type of a program may be inadequate in view of studies demonstrating the prevention of bone atrophy by standing quietly for three hours a day<sup>5</sup>. Under such conditions both the compressional and tensile forces are much greater and for a longer period of time. The type of isotonic leg exercises, the bicycle motion, may not have sufficiently employed the

use of the calf muscles either to properly inhibit the demineralization of the os calcis. Isometric exercises concentrating forces of 300 to 400 lbs. on the os calcis resulted in less of a bone loss, as demonstrated by x-ray scanning of the os calcis. This exercise method also required the use of more leg muscles, particularly in the calf region. The calcium balance data showed no change from untreated bed rested subjects though which would indicate that demineralization of bone must be occurring elsewhere in significant quantities.

The effect of exercise upon bone atrophy in terms of mineral balances and density roentgenograms is still an open issue. Exercises involving important muscle groups, of sufficient duration and force are required to properly evaluate their effect upon bone demineralization. Sensitive measurement techniques are also required in obtaining data on calcium balances, density roentgenograms, and important hormonal changes.

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